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Southwestern MEDICINE

Official Journal of The Southwestern Medical Association,
The Western Association of Railway Surgeons, The Southwest Obstetrical and Gynecological Society,
Southwestern Dermatological Society, Texas District One Medical Association,
The Southwestern New Mexico Medical Society, and El Paso County Medical Society

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VOL. 42, NO. 12

December, 1961

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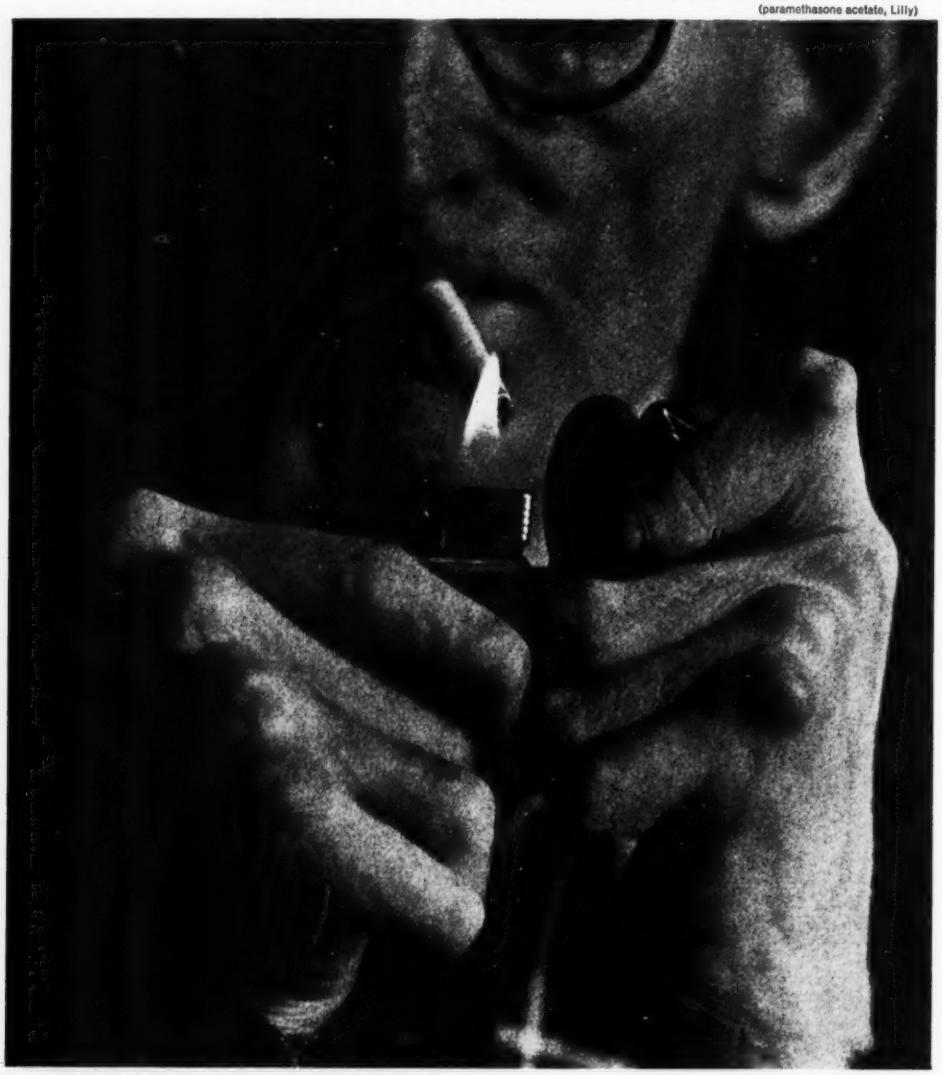

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Located in the heart of the beautiful Phoenix citrus area near picturesque Camelback Mountain, the hospital is dedicated exclusively to the treatment of psychiatric and psychosomatic disorders, including alcoholism.

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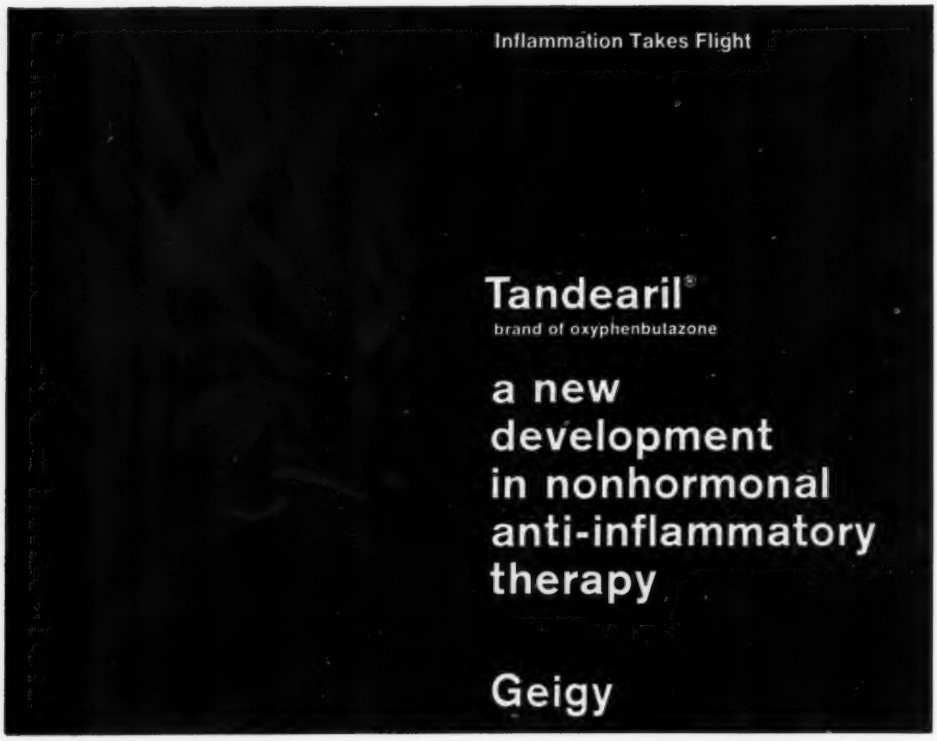

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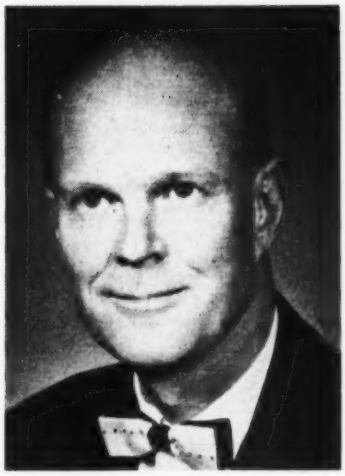
1. Graham, W.: Canad. M. A. J. **82**:1005 (May 14) 1960. 2. Vaughn, P. P.; Howell, D. S., and Kiem, I. M.: Arth. and Rheumat. **2**:212, 1959. 3. O'Reilly, T. J.: J. Irish M. A. **46**:106, 1960. 4. Cardoe, N.: Ann. Rheumat. Dis. **18**:244, 1959. 5. Robichaux, E.: General Practice **24**:14, 1961. 6. Brooke, J. W.: Western Med. **2**:81, 1961. 7. Connell, J. F., Jr., and Rousselot, L. M.: Am. J. Surg. **98**:31, 1959. 8. Brodie, B. B., et al., in Contemporary Rheumatology 1956, p. 600. 9. Stein, I. D.: Ann. N. Y. Acad. Sc. **86**:307 (March 30) 1960. 10. Barczyk, W., and Röth, W.: Praxis **49**:589, 1960. 11. Miller, J. M., et al.: Antibiotic Med. and Clin. Therap. **7**:109, 1960. 12. Connell, J. F., Jr., and Rousselot, L. M.: Am. J. Surg. **97**:429, 1959. 13. Summary of individual case histories submitted to Geigy. 14. Domenjoz, R.: Ann. N. Y. Acad. Sc. **86**:263, 1960. 15. Smyth, C. J.: Ann. N. Y. Acad. Sc. **86**:292, 1960. 16. Yü, T. F., et al.: J. Pharmacol. and Exper. Therap. **123**:63, 1958.

MEETINGS

Dr. Harold J. Beck Elected President of Southwestern Medical Association



Dr. Beck

Dr. Harold J. Beck, Albuquerque, was elected president of the Southwestern Medical Association at its 43rd annual meeting in Las Vegas, Nevada, Oct. 19-21, 1961, at a sparkling convention which brought physicians from as far away as Boston and Honolulu.

Other new officers are Dr. M. D. Thomas, El Paso, president-elect; Dr. Frank A. Shallenberger, Jr., Tucson, vice-president; and Dr. Grady Morrow, El Paso, secretary-treasurer. Members of the executive committee are Dr. Harry W. Sellers, Lordsburg, N. M.; Dr. David Russek, Chihuahua City; Dr. Carlos Sotelo, Hermosillo, Son.; Dr. Louis G. Jekel, Phoenix; and Dr. Louis W. Breck, El Paso, managing editor of *Southwestern Medicine*. Dr. Sherwood Burr, Tucson, was the retiring president.

The 1962 meeting will be held in Albuquerque Oct. 18-20 with headquarters at the Western Skies Hotel.

Over 200 physicians turned out for the annual session which was held at the Tropicana Hotel with such features as the Folies Bergere and Shecky Greene, noted comedian.

Speakers

Speakers were Dr. O. T. Clagett, Head of Section, Division of Surgery at the Mayo Clinic and Professor of Surgery at the Mayo Foundation Graduate School, University of Minnesota; Dr. Arthur C. Curtis, Past President of the American Academy of Dermatology and Syphilology, and Chairman of the Department of Dermatology, University of Michigan Medical Center; Dr. Cary M. Dougherty, Clinical Associate Professor of Obstetrics and Gynecology at the Louisiana State University School of Medicine.

Dr. Max Fine, Associate Clinical Professor of Ophthalmology at the Stanford University School of Medicine and the University of California

Dr. Thomas

Dr. Shallenberger

Dr. Morrow

Medical School; Dr. S. Benjamin Fowler, Associate Professor of Clinical Orthopaedic Surgery at the Vanderbilt University Medical School; Dr. William Parson, Professor of Internal Medicine and Chairman of the Department of Internal Medicine at the University of Virginia School of Medicine; and Dr. Martin Jungmann, Director, Institute for Gravitational Strain Pathology, New York.

Born in Colorado

Born in Los Animas, Colo., the new president, Dr. Beck, received his M.D. from the University of Colorado School of Medicine. He interned in St. Joseph's Hospital in Denver and then took his residency in Urology at the New York Polyclinic Hospital and Christ Hospital in Jersey City.

During World War II he served for two years in the European Theater, emerging with the rank of major. He began the practice of medicine in 1945 in Albuquerque at the Medical Arts Square, still the location of his offices today.

He is a Fellow in the American College of Surgeons, a past president of the Bernalillo County Medical Society, a past chief of staff at St. Joseph's Hospital, and a past president of Surgical Service of New Mexico. He is a member of the Presbyterian Church.

He and Mrs. Beck have a son, John Beck, a sophomore at St. Joseph College and daughter, Mrs. Philip Baiamonte of Albuquerque.

Postgraduate Course Scheduled

Obstetrics and Gynecology will be discussed at a one day course to be given by the El Paso Branch of the University of Texas Postgraduate School of Medicine Sunday, Jan. 21, 1962, in the El Paso County Medical Society's Turner Home at 1301 Montana Avenue in El Paso.

Category I credit for the course has been approved by the Texas Academy of General Practice.

Dr. J. Leighton Green is director of the El Paso Branch.

Dr. Zeph Campbell Heads Southwest OB and Gyn Society

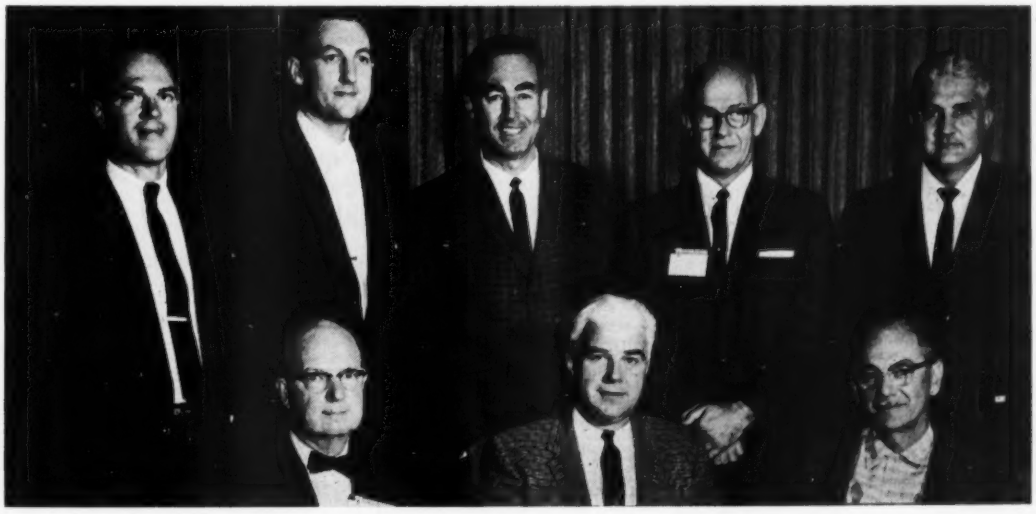
Dr. Zeph Campbell of Phoenix was elected President of the Southwest Obstetrical and Gynecological Society at its eleventh annual meeting in San Diego Oct. 29-31, 1961.

Other new officers are Dr. Hobart Kelly, Riverside, Calif., President-elect; Dr. Max Costin, Tucson, vice-president; Dr. Charles T. Franklin, La Mesa, Calif., secretary; and Dr. Francis L. Rook, San Diego, treasurer. Dr. John F. Wanless of San Diego was the retiring president.

The 1962 meeting will be held at Phoenix, Oct. 10-13, with headquarters at the Camelback Inn.

Guest speakers were Dr. Isadore Dyer, New Orleans, Professor of Obstetrics and Gynecology at Tulane University School of Medicine; Dr. Robert E. L. Nesbitt, Jr., Professor and Chairman of the

SOUTHWEST OB AND GYN OFFICERS—New officers of the Southwest Obstetrical and Gynecological Society, elected at the eleventh annual meeting in San Diego, Oct. 29-31, are, front row (left to right) Dr. Hobart Kelly, Riverside, Calif., President-Elect; Dr. Zeph Campbell, Phoenix, President; and Dr. Max Costin, Tucson, Vice-President. Back row (left to right), Dr. Francis Rook, San Diego, Treasurer; Dr. Charles Franklin, La Mesa, Calif., Secretary; Dr. Charles Weber, La Jolla, Calif., Dr. John Poyas, Laguna Beach, Calif., and Dr. Joseph M. Botte, San Diego, council members.



Department of Obstetrics and Gynecology, State University of New York Upstate Medical Center at Syracuse; and Dr. Buford Word, Birmingham, Professor of Obstetrics and Gynecology at the Medical College of Alabama.

Dr. Ralph A. Reis, Professor of Obstetrics and Gynecology at Northwestern University served as moderator at the luncheon meetings.

Born in Ohio

Dr. Campbell was born in Ada, Ohio, and was reared and attended grade and high school in East Chicago, Indiana .

He then attended Northwestern University for both undergraduate and graduate studies and received his M. D. degree in 1942.

He interned at Passavant Memorial Hospital in Chicago and served his residency at Presbyterian Hospital in Chicago.

For a time he engaged in private practice in Chicago. Then in 1947 he moved to Phoenix and has practiced there ever since.

Railway Surgeons Elect Dr. Hugh S. Collett President

Dr. Hugh S. Collett, Elko, Nev., was elected president of the Western Association of Railway Surgeons at its annual meeting in Reno, Nev., Sept. 28-30, 1961.

Other new officers are Dr. Charles R. Kennedy, Paso Robles, Calif., first vice president; Dr. John C. Mitchell, Salina, Kans., second vice president; Dr. Harry O. Hund, San Rafael, Calif., treasurer; Dr. Graham Owens, Kansas City, secretary; and Dr. Louis E. Jones, Roseville, Calif., past president of the association, chairman of the executive committee.

Speakers at the meeting were Dr. Ruth Fleming, Reno, Surgeon, Western Pacific Railroad; Dr. Maurice E. Leonard, Associate Professor of Clinical Medicine at the University of California and Senior Consulting Internist, Western Pacific Railroad; Dr. Max Childress, Assistant Clinical Professor of Surgery at the University of California and Chief Consulting Thoracic Surgeon, Western Pacific Railroad.

Dr. John M. Read, Elko, Nev.; Dr. Bradford Simmons, San Rafael, Calif.; Dr. Charles J. Monahan, San Francisco; Dr. Angelo Lapi, Professorial Lecturer in Law Medicine at the Kansas City University School of Law and Assistant Professor of Clinical Pathology at the University of Kansas Medical Center.

Dr. H. Corwin Hinshaw, Clinical Professor of Medicine, Stanford University; Dr. Albert C. Daniels, Assistant Clinical Professor of Surgery, Stanford University; Dr. Bernard Kaufman, Sr., Medical Superintendent, Southern Pacific General Hospital; Dr. Emery R. Calovich, Kansas City, Consultant Cardiologist, Union Pacific Railroad; and Mr. Francis B. Lewis, Manager of Safety and Courtesy, Union Pacific Railroad.

Best Papers in Southwestern Medicine To Receive Awards

Five Hundred Dollars to be Presented Annually

A total of Five Hundred ($500.00) Dollars will be awarded annually for the best original scientific articles to be published in *Southwestern Medicine* starting with the January, 1962 issue.

The awards will be made in two classifications: Regional and National.

All physicians who practice in West Texas, Arizona, New Mexico, Nevada or Northern Mexico (States of Sonora and Chihuahua) will be eligible to compete for the Regional Awards.

All physicians in the United States outside the Regional area may compete for the National Awards.

Only original scientific articles published in *Southwestern Medicine* during the calendar year will be eligible.

Awards will be made in the following amounts for the best original articles written by physicians in the Regional Area:

One Hundred ($100.00) Dollars for the best paper.

Seventy-Five ($75.00) Dollars for the second best paper.

Fifty ($50.00) Dollars for the third best paper.

Awards for the best original articles written by physicians in the National classification (outside the Regional Area) will be made in the following amounts:

One Hundred ($100.00) Dollars for the best paper.

Paul I. Murphy (right), president of the Medical Research Association of New York and Boston, presents Dr. Louis W. Breck, managing editor of SOUTHWESTERN MEDICINE with a grant of $500 to launch the journal's Writing Awards contest.

Seventy-Five ($75.00) Dollars for the second best paper.

Fifty ($50.00) Dollars for the third best paper. An additional Fifty ($50.00) Dollars will be set

aside annually to establish a fund for a special classification to be known as the Intern and Resident Writing Awards. Original scientific articles submitted by resident physicians and interns of the Regional Area will be eligible for special awards to be announced at a future date.

The Writing Awards, to be made only for papers published in *Southwestern Medicine*, have been established by Paul I. Murphy, President of Medical Research Association of New York and Boston, to encourage improvement in medical journal writing.

The first annual grant of $500.00 to launch the Writing Awards contest was presented to *Southwestern Medicine* at the annual convention of the Southwestern Medical Association in Las Vegas, Nev. Louis W. Breck, M.D., of El Paso, Managing Editor, accepted in behalf of the Journal.

According to Mr. Murphy, the Writing Awards will be presented annually to focus attention by members of the medical profession on *Southwestern Medicine* as the journal of choice for important contributions. The aim, also, is to emphasize the Southwest Area as a growing clinical research center.

Contributions must be written in English. They must be typed, double spaced and on one side of paper only. A stamped, self-addressed envelope must be included with each paper to insure return of rejected manuscripts.

All papers should be submitted to Lester C. Feener, M.D., Editor, 310 North Stanton Street, El Paso, Texas. As with all official medical journals, only those papers found acceptable by the board of editors of the journal will be published.

Arrangements are being made with the American Medical Writers Association to appoint a panel of judges who will make the annual selection of the best published papers.

ORIGINAL ARTICLES

Acute Transient Diabetes Mellitus with Acidosis and Hypothyroidism

E. W. LANDER, M.D., *Roswell, N. M.*

Acute diabetes mellitus with acidosis complicated by hypothyroidism, hypercholesterolemia, hypertension and obesity can be a stimulating experience to the practicing physician. This is especially true when it all occurs in a patient whose sole complaint was a pruritic dermatitis involving the glans penis and foreskin.

Interest quickens in an attempt to explain the acute culmination of the pathological physiology involved. Could an episode such as this be precipitated by anxiety and the tensions of starting a new business venture?

The following case affords a most interesting interplay of the above-mentioned metabolic dysfunctions. Pertinent facts of the case will be presented. It is proposed to then discuss the separate abnormalities involved and their relation to each other in the clinical picture.

Case Report

A thirty eight year old, obese, white male presented himself in September with the sole complaint of a pruritic balanitis of about two weeks duration. A smear was obtained and culture revealed staphylococcus aureus and monilia albicans. Initial urinalysis revealed an acid urine with a specific gravity of 1.035, no albumin, sugar four plus, acetone four plus and a few hyaline casts.

The history revealed that he had developed obesity and hypertension while serving with the navy during World War II. Blood pressure at that time was said to be as high as 180/120 and the weight up to as much as 260 lbs. He had been given a medical discharge from the Navy because of his hypertension and an anxiety neurosis which was characterized chiefly by severe headaches.

He was told a few years ago, by another doctor, that he had gout, but he had only temporary treatment. He

was now employed as a district salesman, a new position in a new territory. Specific questioning revealed that he had noted polydipsia, polyuria, occasional vertigo and some sub-occipital headaches. He denied any visual disturbance.

He had been taking no corticosteroids or hormones. He was not questioned concerning his sex life or libido. A complete examination at a veterans hospital six months previously was reported by the patient to have revealed no pathology except for the obesity and hypertension which were not treated.

Family history revealed that his obese mother developed diabetes at the age of sixty four. She also had a blood serum cholesterol of 365 mgm. There was a familial tendency to obesity and hypertension, but no known hypothyroidism, goitre, gout or insanity. His wife and sixteen year old daughter each weighed 225 lbs., while two other children were of normal weight.

Pertinent data of the physical examination follows: The eyes and vision were grossly normal. The fundi revealed no unusual abnormality except for the fact that the vessels appeared rather pale and indistinct. The disc margins were normally sharp. The thyroid gland was palpably normal. The heart was negative except for mild general enlargement and the BP was 160/114.

The lungs were clear. The weight was 243 lbs. and the habitus stocky. There was no evidence of arthritis or gouty deposits. The liver was not clinically enlarged. The genitals were negative except for a weeping dermatitis involving the glans and foreskin. There was no edema nor signs of allergy. The hair was prematurely grey. The skin showed no unusual coarseness, xanthoma or xanthelasma.

In addition to the urinalysis noted above, laboratory findings were as follows: Fasting blood sugar 396 mgm.; blood serum cholesterol 1012 mgm.; (the blood serum had the appearance of whipped cream). Blood serum uric acid 4.8 mgm.; protein bound iodine 2.6 gamma; rbc 5,950,000, hb. 18.54, wbc 8,000, seg. 48, non-seg 2, lymphs 40, monos 4 and eosins 6. Unfortunately, determinations of neutral fats and triglycerides were not done at this initial examination; so they were omitted entirely.

Treatment was carried out in the office on an ambulatory basis. He was forbidden to drive his car for the present and activity was limited to the bare essentials of his work.

He was placed on a routine, qualitative reduction diet of low salt and low cholesterol content. NPH Insulin®, U 40, was given each AM at the office with daily urinalysis and fasting blood sugar determinations for the next four days. Dextro amphetamine and amobarbital, 15 mgm. (Dexamyl Spansule®) was prescribed daily AM. The blood sugar at this time was 248 mgm. and the balanitis was practically clear.

Eight days after the onset of treatment the urine was free of sugar, there was only a trace of acetone and the blood sugar was 147 mgm. BP was 140/100 and the weight was 235 lbs. Blood serum cholesterol was 678 mgm. NPH Insulin was reduced to U 20 daily AM and triiodothyronine 5 mcgm. (Cytomel®) daily was started.

Ten days after treatment was started, he suddenly developed a severe visual disturbance which rendered him practically blind. Examination by an ophthalmologist revealed severe hypermetropia which was not properly evaluated at the time. The fundi were said to show no abnormality of note. The vision spontaneously improved during the next week and was again normal in about ten days. The urine was entirely normal and the blood sugar was 128 mgm. three weeks after onset of treatment.

It was decided to change the diabetes therapy from insulin to one of the oral hypoglycemic agents. He was started on 1.0 gm. of tolbutamide (Orinase®) AM and PM. Three days later the urine was still clear and the blood sugar was 117 mgm. The Orinase was reduced to 0.5 gm. AM and PM. Three days later the blood sugar was 107 mgm., BP 140/96, weight 227 lbs. and the vision remained free of defect. The Orinase was reduced to 0.5 gm. daily AM and the Cytomel was increased to 12.5 mcgm. The following week Cytomel was increased to 25 mcgm.

Six weeks after starting treatment the BP was 124/92, weight 222 lbs., urine clear, blood sugar 117 mgm. and blood serum cholesterol 258 mgm. The Orinase was stopped and further treatment consisted only of reduction diet, Cytomel 25 mcgm. and Dexamyl Span. 15 mgm. daily AM. No attempt was made to be more specific with regard to the diet. His obese wife and daughter, who were not examined, cooperated with simultaneous weight reduction. One week later the Cytomel was increased to 50 mcgm. daily.

Approximately eight weeks after the onset of this episode, he developed an acute tonsillitis which was promptly treated with penicillin. This infection failed to cause a flare-up of the diabetes. The patient was followed casually and he checked all urine specimens with Testape®. Four months later the PBI was 3.9 gamma, serum cholesterol 231 mgm., urine clear and the vision normal.

He has remained free of complaint without evidence of return of the diabetes mellitus and has continued to reduce his weight. He has failed to show any clinical or laboratory evidence of gout.

He was changed from Cytomel 50 mcgm. to Proloid® 0.13 gm. with the hope of improving the PBI response. Determination of the latter after two months showed the PBI to be 3.6 gamma; so the patient was returned to the use of Cytomel.

He was followed for one year. Last examination revealed BP 140/96, weight 207 lbs., the urine negative and cholesterol 268 mgm. He was advised to increase the Cytomel to 75 mcgm. daily. The patient moved from this vicinity about this time; so there has been no further follow-up of this case.

Heredity

All the separate pathological entities noted in this case can be attributed to a genetic factor.

The inheritance of diabetes mellitus may be predicted and anticipated according to Mendel's Law.[10]

Hypothyroidism of variable degree has been noted among members of successive generations of the same family.

The obese patient is most often one of a family with a tendency toward excessive weight. On the other hand, environment cannot be disregarded since families live under the same conditions and tend to acquire similar food patterns. Dietary indiscretion throughout childhood and extending into adult life may well be a factor in the development of diabetes, hypertension, arteriosclerosis and hyperlipemia.[6]

Hypertension

A family history of hypertension is more often noted in hypertensive diabetics.[10] It is recognized that the arteriosclerotic changes which develop in later years in the non-diabetic population appear earlier and more frequently in diabetics.[12] Vascular complications have replaced coma as the leading cause of death in diabetes mellitus.

Hypertension may be related to the development of arteriosclerosis due to it increasing the rate of passage of plasma constituents, such as excessive lipo-proteins, into the arterial walls.

Hypotension, rather than hypertension, is more commonly found in the patient with hypothyroidism.

Obesity

The tendency to obesity often begins early in life. Emotional instability is frequently a factor, especially in the obesity of children. The association of obesity with diabetes has been recognized for years. Recent figures[2] indicate that this relationship has risen from 40 percent some fifty years ago to 80 percent at the present time.

Obesity, in some degree, is common but not always an associated finding in patients with hypothyroidism and hypercholesterolemia.[3]

Although obesity is commonly associated with hypertensive cardio-vascular disease,[8] no definite conclusion can be drawn concerning the etiological relationship.

The majority of obese diabetics are adults, usually more than 40 years of age. The disease is less severe when it develops in this age group and the symptoms may be minimal, as shown by this case.

Hypothyroidism

Some clinical reports,[10] in the past, have suggested that hypofunction of the thyroid gland might protect the patient against the development of diabetes mellitus. This conclusion was undoubtedly premature and invalid. Statistical reports are scanty,[13] but there is ample evidence that the two entities do occur simultaneously in the same individual.

Actually, since more precise tests have become available, it has become evident that diabetes mellitus occurs more frequently on the hyperthyroid patient and that diabetics are more apt to develop over-activity of the thyroid gland.[10] Further data indicates that this seems to occur more often on patients with toxic adenoma.

A search of the literature indicates that clinical hypothyroidism[13] and diabetes mellitus seldom develop in the same person. Co-existence of the two entities is more commonly found in the various types of multiglandular disorders, especially those involving the pituitary gland. The role of thyroid hormone in regulation of carbohydrate metabolism is evident from alteration of the latter which occurs with changes in thyroid function. Animal experimentation and clinical observation indicate that alteration of thyroid function results in significant changes in carbohydrate metabolism only when the pancreatic function is impaired.

Rupp, DiGeorge and Paschkis[13] report an interesting case of a 39 year old female with spontaneous myxedema who showed impaired carbohydrate tolerance. She developed clinical diabetes after nine years of treatment with thyroid substance. She stopped thyroid medication and the diabetes subsided, but signs and symptoms of hypothyroidism recurred. She then resumed the thyroid substance and the diabetes returned. The authors concluded that the thyroid hormone merely activated the latent diabetes in this patient rather than actually causing it.

It seems logical to conclude that the administration of thyroid hormone to diabetics who also show evidence of hypothyroidism has the same beneficial effects as it has upon non-diabetics.[14]

It appears to have no effect upon the control of the diabetes.

Hypercholesterolemia

The classification of hyperlipemia into primary and secondary states is perhaps academic and is often difficult to establish clinically. Idiopathic, primary hyperlipemia is said to be characterized by marked elevation of serum triglycerides with a moderate to marked increase of serum cholesterol.[2] The diabetes encountered in this group is usually mild and acidosis seldom occurs.

These cases can frequently be controlled entirely by diet. Xanthomata are common in this condition and are often acute. They tend to clear with improvement of the underlying hyperlipemia. Secondary hyperlipemia is more commonly associated with chronic disease and impairment of kidney function is a fairly constant finding.

It is common knowledge that serum cholesterol is often elevated in the presence of hypothyroidism.[4] Furthermore, the progressive development of arteriosclerosis in the untreated or inadequately treated hypothyroid patient is a major hazard. The hazard is doubled when the patient has a co-existent diabetes, be it frank or latent.

The inefficient or subnormal utilization of carbohydrate[11] as in diabetic acidosis results in fats becoming the prime source of energy. Thus, hyperglycemia is not the determining factor in the production of either diabetic acidosis or hypercholesterolemia. Contrast the hyperlipemia of glycogen storage disease which is characterized by a fasting hypoglycemia. Both diabetes and glycogen storage disease are characterized by inadequate utilization of carbohydrates.

The treatment of diabetes re-establishes adequate utilization of carbohydrates and thus tends to reduce the hyperlipemia and clear the xanthomatoses if present. It seems possible that the coexistence of hypothyroidism may have been overlooked in some of these patients.

Diabetes Mellitus

Diabetes mellitus is a metabolic disease with many facets and possibilities for complications. An attempt will be made to limit the discussion of this condition to the case as presented. The occurrence of diabetic acidosis, even though transient, is closely related to the extreme cholesterolemia, obesity and hypertension noted in this case.

The etiology of acute transient diabetes with acidosis appears to be no different than those factors found in the confirmed diabetic in periods of poor control except that such a patient usually has only a latent diabetes. Again, this is exemplified by this case.

Acidosis and coma are reported to be more common in the summer than in the winter.[10] This may be due to less strict attention to health routine during vacations or possibly related to disturbances in fluid balance.

The hypothyroidism in this case was probably coincidental with respect to the diabetes, but it may well have been a potent factor in regard to the hypercholesterolemia.

Anxiety

The emotional impact of anxiety factors[7] may influence metabolic function in varied fashion. This is typified in disease states of obesity, hypertension, diabetes and even hypothyroidism. Such disturbances can have as deleterious an effect on diabetic control as that associated with infection, starvation, carbohydrate deprivation, immobilization or hyperinsulinism.

Hinkle and Wolf[10] have shown that emotional disturbances in diabetes can produce ketosis and an increased glycosuria which subside with the removal of stress. Danowki[10] reports that 10 to 20 percent of juvenile diabetics admitted to the hospital because of severe acidosis or coma have had a preceding period of emotional turmoil.

Recognition of the fact that emotional disturbances can aggravate pre-existing diabetes makes it logical to assume that they may be responsible for the aggravation of a latent diabetes. Clinical diabetes, thus evolved, is usually labile and transient.

Although such cases have been reported, it is difficult to prove that the diabetes actually originates from stress. Selye has presented us with much scientific data on the subject of stress, but the reaction of the human patient to stress is undoubtedly a highly individual matter.

Lipemia Retinalis

Lipemia retinalis[1] is a rare manifestation of an abnormal concentration of serum lipids most commonly associated with diabetes. It is never seen in the absence of hyperlipemia, but hyperlipemia alone does not produce a milky serum nor the retinalis picture.

These changes are probably due to the abnormally high concentration of the fraction of neutral fats. However, fractionalization of serum lipids yields no practical information not obtainable from a total cholesterol determination. Franklin and Weissmann[1] reported a typical case which also demonstrated xanthomata. The retinal vessels in this case appeared pinkish-yellow and flat due to the turbidity of the serum. There was no arteriolar light reflex; in fact, the arteries and veins could be distinguished only by their size. The fundi were pale, without hemorrhages or exudate and the disc outline sharp.

Heroic treatment of the diabetic acidosis with prompt control resulted in clearing of the retinal vessels by the sixth day. The hyperlipemia cleared by the seventeenth day and the xanthomata disappeared in two months.

The role or possible association of thyroid hormone deficiency in this case was not discussed by the authors.

Transient Changes in Refraction

Transitory refractive changes are not uncommon in diabetic patients, especially the untreated. The sudden onset or increase in myopia suggests inadequate control of the hyperglycemia. Joslin has stated: "The appearance of hypermetropia in a diabetic patient under treatment should suggest too rigorous insulinization." Such changes are transitory and refraction for new or changes of corrective lenses is not necessary.

Stern reports three cases:[9] one of sudden myopia in an untreated case and two of sudden hypermetropia in treated cases. He also presents a brief summation of the theories concerning the etiology and pathological physiology of this condition.

Several explanations have been offered for the transitory ametropia associated with diabetes. All are based on the assumption that the change is due to an increase in the refractive index of the ocular media. There is no agreement, however, on the point at which this increase occurs. The theory ascribing the responsibility for myopia to the presence of an excess of sugar, as such, is untenable for physical reasons. Neither does it explain the changes in astigmatism occasionally observed.

Duke-Elder, in 1943, explained the change in refraction by a disturbance of the osmotic equilibrium between the lens and the aqueous humor. This may be precipitated by sudden change of sugar concentration in the blood. The lens, with its sluggish metabolism, does not participate in this fluctuation of body fluids and tries to maintain its original molecular concentration. The osmotic equilibrium is maintained by the entrance of fluid into the lens from the aqueous humor. This causes the lens to swell and its surface to adapt to the increased curvature.

In addition, the optic density of the peripheral layers decreases while that of the nucleus remains unchanged. These factors cause the refractive power of the lens to increase and the eye to become myopic. Should the blood sugar suddenly decrease, as occurs in the vigorous treatment of diabetic acidosis, the opposite mechanism functions to produce hypermetropia.

Management

The treatment of the obese diabetic should be directed primarily to the obesity. In fact, it is apparent that weight reduction should be advised for the correction of all of the pathological disturbances involved in this case.

The use of otherwise adequate low fat diets in the therapy of diabetes mellitus deserves reemphasis. On the contrary, the use of the old fashioned ketogenic diet is no longer justified because of the probable relationship between high caloric, high fat diets and atherogenesis. The treatment and correction of diabetes per se will usually have little effect on an associated hyperlipemia.

The National Institute of Health reports some recent enzyme research which suggests that intravascular clotting is more likely to occur after a fatty meal. The frequent association of hypercholesterolemia and ateriosclerosis has long been recognized; although the exact role of cholesterol is not clear. Pathological reports indicate that arteriosclerosis is a disease of progression. The initial lesions have been noted even in infancy. Insull and Ahrens[16] have shown that the highly saturated fatty acid content of the American diet is even reflected in the high level of lipids in breast milk.

A large number of studies substantiate the fact that polyunsaturated fatty acids tend to reduce

the serum level of cholesterol; whereas, a high intake of saturated fats produces the reverse. The restriction of certain foods of high cholesterol content has been carried to extremes in the attempt to treat obesity and hypercholesterolemia. Eggs, for instance, almost always prohibited, have far too many other desirable food qualities to be placed on these lists of forbidden foods.

The oral hypoglycemic agents have proven valuable and facilitated the treatment of certain types of diabetes mellitus, especially in the obese adult. New thyroid analogues may prove to be the answer in the search for specific blood-cholesterol-lowering agents according to Corday.[15] The calorigenic effect of these chemicals seems to be minimal; whereas, the cholesterol-lowering effect is strong. Toxic effects have been few and reversible in controlled studies.

The role of thyroid hormone in cholesterol metabolism is thus given additional emphasis. However, Bissell states that the PBI determination cannot be used as an index of therapeutic efficacy when Cytomel is used to treat the hypothyroid state. The clinical response to Cytomel in the reported case was much greater than the PBI determinations would indicate.

The use of anorectics has been popularized as an adjuvant for the reduction of excess weight. It seems that the value of these agents has been overly rated; although, they may have a temporary deterrant effect on the appetites of some patients.

Discussion

This case appears to be that of a latent diabetic with multiple metabolic disturbances. The cause of the acute diabetic acidosis is conjectural. It may well have been precipitated by anxiety and stress.

The associated pathological physiology secondary to the obesity, hypertension, hypothyroidism and hypercholesterolemia was, no doubt, instrumental in the development of the diabetic crisis. The hypothyroidism had probably been present for years and is coincidental with regard to the diabetes. It could, however, have a direct effect on the severity of the hypercholesterolemia. The development of diabetic acidosis in such a case is reportedly rare and is usually not severe.

The transient abnormalities involving the eyes, such as lipemia retinalis and acute refractive changes, are worthy of emphasis. The literature is replete with the retinal pathology associated with diabetes, but it is rare to find these two complications even mentioned.

Summary

A case of acute transient diabetes with acidosis has been presented and discussed. The occurrence of this condition in a patient with hypothyroidism is not common. The initial manifestation and sole complaint was a pruritic balanitis. Further study disclosed multiple metabolic disturbances of unusual interest. The medical care of this case was rendered without hospitalization or loss of time from employment.

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Combined Therapy In the Treatment of Hypertension

Reserpine, Hydralazine, Hydrochlorothiazide

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Essential hypertension is a medical enigma. Although some cases of arterial hypertension are the result of renal disease or hyperadrenocorticism and are so diagnosed, it still remains that the majority of patients fall into the reservoir of essential hypertension . . . a state where the etiology may be quite obscure.

Increase in peripheral resistance and elevated blood pressure is a result of many factors. It has been suggested that the essential cause of the increased peripheral resistance is an inherent hypersensitivity of the vasoconstrictor nerves and an exaggeration of the usual vasomotor reflexes. These factors tend to increase the neurogenic impulses or the humoral mechanisms when the factors which regulate the receptivity of the vessel walls remain intact.[1]

The nervous and emotional strains of our modern society are underlying factors which lead to essential hypertension by constantly increasing neurogenic impulses. While kidney disease is not a precursor to essential hypertension it becomes a sequel to it by reducing renal blood flow.[1]

There is little doubt that disturbances in biochemical and neuro-biochemical mechanisms are forerunners of essential hypertension, yet these areas of physiological imbalance are most difficult to diagnose.[2] About 75 per cent of all patients fall into this category.[2]

Therapeutic Agents

Since the etiology of essential hypertension is obscure, complex, and practically defies accurate diagnosis, it follows that therapeutic measures must be aimed at treating symptoms. Naturally, if the pathogenesis could be easily determined then empirical treatment would be unnecessary and indefensible.[3,4,5] The practical result of these circumstances is that all patients receive a series of therapeutic agents or a prescribed mixture of drugs. Hypertensives with a history of nervous and emotional stress are treated with sedatives and calming agents with considerable success.

One of the most effective of these is reserpine (Serpasil®). Many clinical studies have demonstrated that reserpine acts as a shield against stress by preventing responses to excessive psychic stimulation. [6,7,8,9] Compounds of the phthalazine group have hypotensive properties. One of these, hydralazine (Apresoline®), has a remarkably long-lasting vaso-pressor effect, gives a rise in pulse rate and an increase in renal blood flow.[10,11,12,13,14]

A combination of reserpine and hydralazine has proved to be an effective anti-hypertensive agent.[15,16,17] During the past few years hydrochlorothiazide (Esidrix®), the natriuretic agent, has also proved most satisfactory in the treatment of hypertension.[18,19,20,21,22]

Therapeutic Rationale

The concept of using several antihypertensive agents in combination drug therapy has received attention from investigators.[23,24] It is known that the thiazide derivatives exert a modest hypotensive action on their own even in the absence of edema. Furthermore, they enhance the potency of other antihypertensive agents such as reserpine and hydralazine.

When one drug potentiates the other it is possible to achieve good therapeutic results with a combination containing smaller doses of each of the drugs. Consequently, the total amount of medication required for effective therapy will be less than when these agents are given alone or in sequence.

After careful review of pertinent data on the hypertensive agents reserpine, hydralazine and hydrochlorothiazide, a preparation was selected for study. This product contained reserpine 0.1 mg., hydralazine 25 mg., and hydrochlorothiazide 15 mg. (Ser-Ap-Es®)*.

Subjects and Methods

Forty patients were used in this study. Selection was made on the basis of blood pressure readings taken several months prior to the investigation. The highest reading was 300/280 mm. Hg. the lowest reading was 180/110 mm. Hg. During the test period, routine pulse and blood pressure readings were taken on each visit. A urinalysis was done on each patient visit.

Patients ranged in age from 29 to 72 years. Fourteen were from 60 to 72 years old. Twelve were 50 to 58 years old. The balance of fourteen were 29 to 49 years of age. Twenty were obese; five were diabetics; three were neurotics, and one had arteriosclerotic heart disease. Six patients had renal hypertension, while the balance of 34 had essential hypertension ranging from moderate to severe. The general clinical condition of all patients has been summarized on Chart No. 1.

The period of treatment ranged from two to fourteen weeks with an average of 4.97 weeks. The combination of drugs was administered in the form of tablets and ranged from one-quarter to four per day. (Chart No. 2 and No. 4)

It is a well known fact that many older people succumb to cerebral accidents and allied vascular disorders related to their hypertension. In these patients, a diastolic blood pressure below 95 mm. Hg. is often a precursor to cerebral myocardial or renal ischemia. Therefore, patients in the age group of 50 to 72 years were placed in a separate category, and therapy was discontinued when the diastolic blood pressure reached the critical range.

Clinical Results

Group 1—Patients Aged 50-72 Years

Three patients in this group were seventy-two years old. Patient No. 1 demonstrated, on one tablet per day, an excellent response. His blood pressure went from 240/180 mm.Hg. down to 150/80 mm.Hg., with a drop in the mean arterial blood pressure of 95* mm.Hg. Patient No. 2, also seventy-two years old, went from 300/150 mm.Hg. to 160/80 mm.Hg., with a reduction in the mean arterial blood pressure of 105 mm.Hg. This is a significant clinical response on only two tablets per day for a two-week period. Previous medications had no beneficial effects upon this patient's hypertension.

Patient No. 3, aged seventy-two, received medication for two weeks with an initial dose of one tablet per day and a dosage reduction to one-half tablet daily. Her blood pressure dropped from 260/160 mm.Hg. to 160/84 mm.Hg., with a reduction in the mean arterial blood pressure of 88 mm.Hg. There were no untoward reactions in any of these three difficult, elderly patients.

The remainder of the patients in Group One were aged 50 to 66 years. They received medication for periods ranging from two to fourteen weeks. One patient, No. 7, required fourteen weeks before a satisfactory clinical response could be achieved. Dosage ranged from one-quarter tablet per day to three tablets per day, with a daily average of two tablets.

Both Patient No. 4 and No. 12 showed marked improvement. The blood pressure readings of Patient No. 12 went from 300/160 mm.Hg. to 160/90 mm.Hg., resulting in a drop in mean arterial blood pressure of 105 mm.Hg. Patient No. 4 went from 280/180 mm.Hg. to 140/94 mm.Hg., with a drop in the mean arterial blood pressure of 113 mm.Hg.!

Patient No. 16 had previously been uncontrolled on various other medications, yet the preparation under study brought her blood pressure down from 220/130 mm.Hg. to 180/100 mm.Hg.; her decrease in mean arterial blood

*The Ser-Ap-Es® used in this study was supplied through the courtesy of Robert D. Graupner, M. D., of Ciba Pharmaceuticals, Inc., Summit, New Jersey.

*Calculated by the method of Best and Taylor: The Physiological Basis of Medical Practice, 6th Ed., Williams & Wilkins Co., Pub. 1955.

Chart No. 1

Summary of Patients' Clinical Status

Case No.	Patient	Age	Sex	Blood Pressure Before	Blood Pressure After	Primary Diagnosis	Secondary Diagnosis	Duration of Disease	Pulse Before	Pulse After	Lab. Data	Weight Before	Weight After	Diet
1	C.I.	72	M	240/180	150/80	EH-S	As.H.D.	10 yrs.	80	80	Neg	167	165	—
2	M.A.	72	F	300/150	160/80	EH-S	Neurotic	Years	84	80	Neg	176	176	—
3	H.L.	72	F	260/160	160/84	EH-S	C.H.D.	Years	86	80	Neg.	116	116	—
4	D.B.	66	F	280/180	140/94	EH-S	Mk.Obes.	Years	90	76	Neg.	250	239	Reducing
5	F.B.	66	M	260/140	180/90	RH-S	Obesity	Years	80	66	2+Alb. to Neg.	170	165	—
6	E.C.	65	F	280/120	150/80	EH-S	Diabetes	Years	80	80	Neg.	173	173	Diabetic
7	M.M.	65	F	260/140	180/90	EH-S	Obesity	10 yrs.	80	64	Neg.	160	155	—
8	G.D.	65	M	200/110	140/80	EH-M	As.C.	Years	80	68	Neg.	141	130	—
9	H.R.	63	F	200/100	180/100	EH-S	Obesity	6+ yrs.	72	80	3+Alb. to Neg.	220	216	Reducing
10	K.T.	62	F	220/120	130/80	EH-M	Mk.Obes.	Years	78	72	2+Alb. to Neg.	194	194	Reducing
11	F.Q.	60	F	300/164	200/110	EH-S	Obesity	Years	86	86	Neg.	220	220	Reducing
12	L.L.	60	F	300/160	160/90	EH-S	Obesity	Years	80	60	Neg.	174	172	Reducing
13	W.I.	60	F	240/140	180/90	RH-S	Obes.&As.	Years	74	74	2+Alb. to Neg.	150	145	Reducing
14	C.J.	60	F	280/140	160/84	RH-S	Obesity	Years	90	84	Neg.	300	300	Reducing
15	L.W.	58	M	220/110	130/80	EH-S	C.H.D.	3+ yrs.	80	80	1+Alb. to Neg.	165	150	Low Na
16	J.B.	57	F	220/130	180/100	EH-M	Neurosis Anxiety	10 yrs.	86	84	Neg.	178	178	Low Prot
17	W.W.	56	F	220/110	170/90	EH-M	Diabetes, Obesity	Years	80	78	Neg.	170	170	Diabetic
18	E.T.	56	F	300/140	160/90	EH-S	Obesity	Years	82	80	Neg.	204	204	Reducing
19	J.S.	56	M	220/136	180/90	EH-M	Arthritis, Obesity	Years	80	74	Neg.	200	194	Reducing
20	N.S.	55	F	300/160	180/100	EH-S	Diabetes	Years	84	80	Neg.	165	165	Diabetic
21	V.H.	50	F	260/150	160/90	EH-S	Obesity	Years	100	82	Neg.	172	163	Reducing
22	S.J.	54	F	180/110	120/80	EH-S	Obesity	Years	80	64	Neg.	245	227	Reducing
23	L.H.	50	F	200/100	180/100	EH-M	—	Years	45	70	Neg.	143	143	—
24	T.M.	50	M	200/100	150/90	RH-M	—	Recent	100	88	4+Alb.	172	171	Na Free
25	W.B.	50	F	240/140	170/90	EH-S	C.H.D.	15 yrs.	86	74	Neg.	190	180	Low Na
26	T.R.	50	F	220/120	160/90	EH-M	Obesity	Years	80	74	Neg.	220	218	Reducing
27	E.M.	48	M	200/106	160/110	EH-M	—	Years	80	80	Neg.	185	185	Reducing
28	R.W.	48	F	220/120	180/100	EH-S	Diabetes	Years	120	86	Neg.	161	160	Diabetic
29	L.R.	47	F	300/280	140/80	EH-S	Obesity	Years	88	80	Neg.	250	250	Reducing
30	V.A.	47	F	180/110	130/80	RH-M	Diabetes	Years	86	80	2+Alb. to Neg.	154	150	Diabetic
31	G.C.	46	M	240/130	140/96	EH-S	—	Years	70	74	Neg.	193	182	—
32	J.T.	45	M	200/120	160/100	EH-M	—	6+ yrs.	72	74	Neg.	172	180	—
33	W.G.	45	M	260/140	160/90	EH-S	—	10 yrs.	80	80	Neg.	230	230	Reducing
34	E.H.	44	F	260/140	130/80	EH-M	Obesity	Recent	80	70	Neg.	225	220	Reducing
35	W.O.	42	M	200/120	120/80	EH-M	Obesity	4 yrs.	82	80	Neg.	215	210	Reducing
36	B.C.	40	F	200/120	150/90	EH-M	Obesity	Recent	88	80	Neg.	225	221	Reducing
37	L.W.	36	F	260/150	180/110	EH-M	Neurosis	Years	80	88	Neg.	182	182	Reducing
38	F.L.	33	F	200/120	140/80	EH-M	—	Years	88	80	1+Alb. to Neg.	114	114	—
39	M.W.	32	F	200/130	160/96	RH-S	Obesity	Years	90	85	2+Alb. to Neg.	195	178	Reducing
40	A.D.	29	F	240/160	150/90	EH-S	Obesity	Years	80	80	Neg.	210	210	Reducing

	Before mm. Hg.	*After mm. Hg.*	*Decrease mm. Hg.*
Average Systolic Blood Pressure	239	157	82
Average Diastolic Blood Pressure	137	90	47

Key to Symbols

E.H.	Essential Hypertension	Mk.Obes.	Marked Obesity
S	Severe	R.H.	Renal Hypertension
M	Moderate	C	Cerebral
As.H.D.	Arterioslerotic Heart Disease	C.H.D.	Coronary Heart Disease

pressure was 35 mm.Hg. Patient No. 22 had been given every known antihypertensive agent and still remained uncontrolled. She was given the reserpine, hydralazine, hydrochlorothiazide compound, one tablet per day for six weeks and then one-half tablet per day for three weeks. Her reading went from 180/110 mm.Hg. to 120/80 mm. Hg., with a corresponding reduction of 45 mm. Hg. in the mean arterial blood pressure.

Similarly, Patient No. 25, who had never been controlled by many other antihypertensives, went from 240/140 mm.Hg. to 170/90 mm.Hg.; the reduction in her mean arterial blood pressure was 60 mm.Hg. Her initial dose was one tablet daily, which was gradually increased over a six week span to three tablets per day. At the end of this period of six weeks, her hypertension was under control.

Patient No. 18 acquired severe dizziness and headache on one tablet three times a day yet, when the dosage was reduced, all symptoms that might have been construed as side-reactions to the drug disappeared. Her original reading was 300/140 mm.Hg. which, in a period of two weeks, was reduced to 160/90 mm.Hg. Therefore, therapy was discontinued for her mean arterial blood pressure had gone from 220 mm.Hg. to 125 mm.Hg.—a reduction of 95 mm.Hg.

Therapeutic results in Patient No. 5 were excellent, although he complained of slight dizziness upon standing. His readings were 260/140 mm.Hg. down to 180/90 m.Hg., and the mean arterial blood pressure dropped from 200 mm.Hg. to 135 mm.Hg., a reduction of 65 mm. Hg. Patient No. 6 complained of slight nausea when tablets were taken before meals. The drug was subsequently taken after meals and she no longer complained. Her readings were 280/120 mm.Hg. down to 150/80 mm.Hg., a reduction of 85 mm.Hg. in the mean arterial blood pressure.

Patient No. 26 complained of lassitude and weakness in the limbs. This was probably due to a prior rigid reducing diet rather than the drug. Her blood pressure dropped from 220/120 mm. Hg. to 160/90 mm.Hg., and the mean arterial blood pressure went from 170 mm.Hg., to 125 mm.Hg., a reduction of 45 mm.Hg. (Charts No. 2 and No. 3)

When this investigation started, the average mean arterial blood pressure in Group 1 was 191 mm.Hg. When therapy was discontinued, the average mean arterial blood pressure was 126 mm.Hg. This represents an average decrease of 65 mm.Hg. in the mean arterial blood pressure for this group. (Chart No. 3)

When the diastolic pressure dropped below 95 in this group, it was the indication for the discontinuance of medication. Previous to treatment the average diastolic pressure was 135 mm.Hg. and when therapy ceased the average diastolic pressure was 89 mm.Hg.

Group 2—Patients Aged 29 to 48 Years

In this category there was a truly amazing therapeutic result. Patient No. 29 began treatment under reserpine, hydralazine and hydrochlorothiazide with blood pressure readings of 300/280 mm.Hg. After two weeks of therapy on one tablet twice a day, his blood pressure was reduced to 140/80 mm.Hg. This represents a drop in mean arterial blood pressure of 180 mm. Hg., for it went from 290 mm.Hg. to 110 mm.Hg.

Patients No. 28, 32 and 33 had never been controlled before on other medications. After six weeks of treatment the blood pressure of Patient No. 28 was reduced from 220/120 mm.Hg. to 180/100 mm.Hg.—a reduction in the mean arterial blood pressure from 170 mm.Hg. to 140 mm.Hg., or a decrease of 30 mm.Hg.

Patient No. 32 was treated for five weeks. His blood pressure went from 200/120 mm.Hg. to 160/100 mm.Hg.; a drop in the mean arterial blood pressure of 30 mm.Hg. since it went from 160 mm.Hg. down to 130 mm.Hg. Patient No. 33 required only two weeks of therapy in order to reduce his blood pressure from 260/140 mm. Hg. to 160/90 mm.Hg. This represents a drop of 75 mm.Hg. in the mean arterial blood pressure.

Ganglionic blocking agents were previously required to control the hypertension in Patient No. 39. She required very careful supervision for, in addition to her severe renal hypertension, she had twice been an eclamptic. Therapy began with one tablet three times a day; at the beginning of the fourth week dosage was increased to two tablets twice a day and at the start of the sixth

Chart No. 2

Group 1—Patients 50 to 72 Years Old—Clinical Response Following Therapy

Case No.	Patient	Sex	Age	Blood Pressure Before Therapy	Blood Pressure After Therapy	Drug Dosage	Length of Therapy	Clinical Condition After Therapy	Comment
1	C.I.	M	72	240/180	150/80	Started 1 per day, increased to 1 B.I.D.	5 weeks	Dizziness and headache gone.	Fine response in elderly patient.
2	M.A.	F	72	300/150	160/80	1 B.I.D.	2 weeks	Headache gone.	No response from other medications. This medication produced significant response in short period.
3	H.L.	F	72	260/160	160/84	Started 1 per day, decreased to ½.	2 weeks	Dizziness gone.	Excellent on small dosage.
4	D.B.	F	66	280/180	140/94	Started 1 T.I.D., increased to 1 Q.I.D.	8 weeks	Marked obesity, but dizziness gone.	Significant response to therapy.
5	F.B.	M	66	260/140	180/90	Started 1 per day, increased to 1 T.I.D.	5 weeks	Feels much better, but complains on standing.	Response to therapy excellent.
6	E.C.	F	65	280/120	150/80	1 Tab B.I.D. to ½ Tab. per day.	2 weeks	Some nausea.	Excellent response, but 6 weeks after therapy blood pressure 240/120.
7	M.M.	F	65	260/140	180/90	1 Tab T.I.D. to ½ Tab daily.	14 weeks	Patient feels fine.	This patient took longer to respond, but clinical result is excellent.
8	G.D.	M	65	200/110	140/80	1-½ daily to ½ daily.	6 weeks	Patient feels fine.	Significant therapeutic response.
9	H.R.	F	63	200/100	180/100	2 Tabs daily.	7 weeks	Patient symptom-free.	Good therapeutic response. Perhaps larger dosage should have been given to reduce time.
10	K.T.	F	62	220/120	130/80	1 Tab daily to ¼ daily.	5 weeks	Exertional dyspnea.	Therapy discontinued when blood pressure dropped to this level. However, response to drug combination excellent.
11	F.Q.	F	60	300/164	200/110	1 Tab daily to 2 Tabs daily.	2 weeks	Patient symptom-free.	Significant therapeutic response.
12	L.L.	F	60	300/160	160/90	1 T.I.D. to 1 daily.	7 weeks	Patient free from dizziness.	Significant therapeutic response.
13	W.I.	F	60	240/140	180/90	1 T.I.D.	14 weeks	Patient free from headache and dizziness.	Significant therapeutic response.
14	C.J.	F	60	280/140	160/84	1 daily	2 weeks	Patient symptom-free.	Response excellent. Surgery required, therapy discontinued.
15	L.W.	M	58	220/110	130/80	1 T.I.D. to 1 daily.	7 weeks	Patient symptom-free.	Significant therapeutic response.
16	J.B.	F	57	220/130	180/100	1 B.I.D.	2 weeks	Tense patient.	All previous therapy without effect. Response to this medication was excellent.

CHART No. 2—*Continued*

Case No.	*Patient*	*Sex*	*Age*	*Blood Pressure Before Therapy*	*Blood Pressure After Therapy*	*Drug Dosage*	*Length of Therapy*	*Clinical Condition After Therapy*	*Comment*
17	W.W.	F	56	220/110	170/90	1 Tab daily.	6 weeks	Patient symptom-free.	Significant therapeutic response.
18	E.T.	F	56	300/140	160/90	1 T.I.D. to 1 B.I.D.	2 weeks	Patient symptom-free.	Significant therapeutic response.
19	J.S.	M	56	220/136	180/90	1 Tab daily to ½ Tab daily.	3 weeks	Patient symptom-free.	Significant therapeutic response.
20	N.S.	F	55	300/160	180/100	1 Tab daily to 1-½ Tabs daily.	6 weeks	Patient symptom-free.	Significant therapeutic response.
21	V.H.	F	50	260/150	160/90	1 B.I.D. to 1 Tab daily.	5 weeks	Patient fine, symptom-free.	Significant therapeutic response.
22	S.J.	F	54	180/110	120/80	1 Tab per day to ½ Tab daily.	9 weeks	Patient symptom-free.	This patient previously received every known type of therapy without results. This combination has been significantly successful.
23	L.H.	F	50	200/100	180/100	1 T.I.D. to ½ Tab daily.	6 weeks		Patient has bradycardia. At one point pulse dropped to 45 up to 60—presently 70. Very uncooperative.
24	T.M.	M	50	200/100	150/90	1 Tab daily.	2 weeks	Patient symptom-free.	Significant therapeutic response.
25	W.B.	F	50	240/140	170/90	1 T.I.D. increased from 1 daily.	6 weeks	Patient symptom-free.	This patient was *not* previously controlled on any type of therapy. This combination has been significantly successful.
26	T.R.	F	50	220/120	160/90	1 Tab daily.	2 weeks	Patient feels better. Complains of weakness. Weighs 218 lbs.	Significant therapeutic response.

Each Tablet Contains: Reserpine 0.1 mg., Hydralazine 25 mg. and Hydrochlorothiazide 15 mg.

N.B.: Under column "Clinical Condition After Therapy," the remark "Patient Symptom-Free" refers to hypertensive symptoms *only*, and does not refer to the patient's secondary disease when such is present.

week, until the end of her course, she received one tablet daily. The result—blood pressure 200/130 mm.Hg. down to 160/96 mm.Hg., or the mean arterial blood pressure reduced by 37 mm. Hg. from 165 mm.Hg. to 128 mm.Hg.

Drug dosage for Patient No. 38 was reduced from one tablet twice a day to one-quarter tablet per day. On this very small dose her blood pressure went from 200/120 mm.Hg. to 140/80 mm. Hg. which represented a reduction of 50 mm. Hg. in the mean arterial blood pressure.

Patient No. 36 complained of dizziness during therapy. The medication was discontinued for one week and then resumed. During the balance of treatment, there were no reactions.

At the beginning of therapy for Group 2, the average mean arterial blood pressure was 182 mm.Hg. At the end of therapy, the average mean arterial blood pressure was 120 mm.Hg., representing an average decrease of 62 mm.Hg. (See Charts No. 4 and No. 5)

Discussion

When this investigation began it was believed that a reduction of 20 mm.Hg. in the mean arterial blood pressure would represent a significant therapeutic response, especially among the

CHART NO. 3

Group 1—Patients 50-72 Years Old

Decrease In Mean Arterial Blood Pressure

Case No.	Patient	Sex	Age	Mean Arterial Blood Pressure Before Therapy mm. Hg.	After Therapy mm. Hg.	Decrease mm. Hg.
1	C.I.	M	72	210	115	95
2	M.A.	F	72	225	120	105
3	H.L.	F	72	210	122	88
4	D.B.	F	66	230	117	113
5	F.B.	M	66	200	135	65
6	E.C.	F	65	200	115	85
7	M.M.	F	65	200	135	65
8	G.D.	M	65	155	110	45
9	H.R.	F	63	150	140	10
10	K.T.	F	62	170	105	65
11	F.Q.	F	60	232	155	77
12	L.L.	F	60	230	125	105
13	W.I.	F	60	190	135	55
14	C.J.	F	60	210	122	88
15	L.W.	M	58	165	105	60
16	J.B.	F	57	175	140	35
17	W.W.	F	56	165	130	35
18	E.T.	F	56	220	125	95
19	J.S.	M	56	178	135	43
20	N.S.	F	55	230	140	90
21	V.H.	F	50	205	125	80
22	S.J.	F	54	145	100	45
23	L.H.	F	50	150	140	10
24	I.M.	M	50	150	120	30
25	W.B.	F	50	190	130	60
26	T.R.	F	50	170	125	45

Average Mean Arterial Blood Pressure At Beginning of Therapy.....191 mm.Hg.

Average Mean Arterial Blood Pressure At End of Therapy.....126 mm.Hg.

Average Decrease In Mean Arterial Blood Pressure..... 65 mm.Hg.

* Mean Arterial Blood Pressure = $\frac{\text{Systolic} + \text{Diastolic}}{2}$

* Calculated by the method of Best and Taylor: The Physiological Basis of Medical Practice. 6th Ed., Williams & Wilkins Co., Pub. 1955.

fragile, older patients. As the study proceeded, it was demonstrated that the original estimate of reduction in the mean arterial blood pressure was too conservative.

Among the older patients, three, all over 60 years of age, had a decrease of over 100 mm.Hg. in their mean arterial blood pressure without any symptoms of cerebral or myocardial ischemia. (See Charts No. 2 and No. 3—Cases No. 2, No. 4 and No. 12) The patients in Group 1, aged 50 to 72, had an average reduction in their mean arterial blood pressure of 65 mm.Hg. (See Chart No. 3).

Based upon this study, it is probable that a reduction of 60 mm.Hg. in the mean arterial blood pressure could represent a highly significant response to the reserpine, hydralazine, hydrochlorothiazide compound. It was also believed that the reductions in the mean arterial blood pressures among the younger group of patients (Group 2, aged 29 to 48) would be greater.

A glance at Chart No. 5 demonstrates that the average reduction in the mean arterial blood pressures among these hypertensives was 62 mm. Hg. It is therefore very probable that when the therapeutic agent under study is carefully used for a sufficient length of time, with proper dosage adjustment, a reduction of 60 mm.Hg. in the mean arterial blood pressure could be considered significant.

The seeming evidence of synergism of these drugs is overwhelming. When reserpine and hydralazine are given together an average daily dose is reserpine 0.8 mg., hydralazine 300 mg. A good average daily dose of hydrochlorothiazide is 100 mg. The average dose of the drug compound used in this study, two tablets per day, represents the daily administration of reserpine 0.2 mg., hydralazine 50 mg. and hydrochlorothiazide 30 mg.

In other words, two tablets of this compound represents only 25 per cent of the ordinary daily dose of reserpine, 17 per cent of the ordinary daily dose of hydralazine, and 33 per cent of the average daily dose of hydrochlorothiazide—yet it is possible with this compound to achieve more than satisfactory therapeutic effects with minimal side reactions.

Summary

Forty hypertensive patients ranging in age from 29 years to 72 years of age were treated with a compound containing per tablet: reserpine 0.1 mg., hydralazine 25 mg. and hydrochlorothiazide 15 mg. The average length of treatment lasted 5.02 weeks. The average daily dose was two tablets. The decrease in systolic blood pressure in Group 1 (50 to 72 years of age) averaged 86 mm.Hg., and in Group 2 (29 to 48 years of age) the average decrease was 78 mm.Hg.

The average decrease in diastolic blood pressure in Group 1 was 46 mm.Hg., and in Group 2 it was 48 mm.Hg. The Group 1 average decrease in mean arterial blood pressure was 65 mm. Hg., and in Group 2 it was 62 mm.Hg.

Chart No. 4

Group 2—Patients 29 to 48 Years Old—Clinical Response Following Therapy

Case No.	Patient	Sex	Age	Blood Pressure Before Therapy	Blood Pressure After Therapy	Drug Dosage	Length of Therapy	Clinical Condition After Therapy	Comment
27	E.M.	M	48	200/106	160/110	1 Tab daily down to ½ Tab daily.	6 weeks	Patient symptom-free.	Good therapeutic results.
28	R.W.	F	48	220/120	180/100	1 Tab daily to 1 B.I.D.	6 weeks	Patient symptom-free.	This patient never controlled before. Excellent response.
29	L.R.	F	47	300/280	140/80	1 B.I.D.	2 weeks	Patient symptom-free.	This is an amazing therapeutic result. Mean arterial blood pressure down 180. (See Chart No. 5)
30	V.A.	F	47	180/110	130/80	1 Tab daily up to 1 B.I.D.	5 weeks	Patient symptom-free.	Good therapeutic response.
31	G.C.	M	46	240/130	140/96	1 Tab daily up to 1 T.I.D.	5 weeks	Patient symptom-free.	Good therapeutic response.
32	J.T.	M	45	200/120	160/100	1 Tab daily.	5 weeks	Patient tense but symptom-free.	Significant therapeutic response. Never controlled on other medication.
33	W.G.	M	45	260/140	160/90	2 T.I.D. up to 5 Tabs daily.	2 weeks	Patient symptom-free.	Significant therapeutic response. Patient has never before been controlled on other medications.
34	E.H.	F	44	260/140	130/80	1 Q.I.D. down to 1 Tab daily.	3 weeks	Patient symptom-free.	Significant therapeutic response.
35	W.O.	M	42	200/120	120/80	1 Tab daily down to ½ Tab per day.	3 weeks	Patient symptom-free.	Significant therapeutic response.
36	B.C.	F	40	200/120	150/90	1 Tab daily up to 1 B.I.D.	7 weeks	Patient complained of dizziness.	Significant therapeutic response. Reduced dosage would eliminate possible reaction.
37	L.W.	F	36	260/150	180/110	1 Tab B.I.D.	2 weeks	Patient symptom-free.	Significant therapeutic response.
38	F.L.	F	33	200/120	140/80	1 B.I.D. down to ¼ Tab daily.	4 weeks	Patient symptom-free.	Significant therapeutic response on an extremely small dose of drug.
39	M.W.	F	32	200/130	160/96	2 Tabs B.I.D. down to 1 Tab daily.	10 weeks	Patient symptom-free.	Good therapeutic response. Patient previously controlled on ganglionic blocking agents.
40	A.D.	F	29	240/160	150/90	1 Tab daily.	2 weeks	Patient symptom-free.	Significant therapeutic response.

Each Tablet Contains: Reserpine 0.1 mg., Hydralazine 25 mg. and Hydrochlorothiazide 15 mg.

N.B.: Under column "Clinical Condition After Therapy," the phrase "Patient Symptom-Free" refers *only* to hypertensive symptoms and does not refer to the patients other disease(s) when one or more exists. (See Chart No. 1)

CHART No. 5

Group 2—Patients 29-48 Years Old

Decrease In Mean Arterial Blood Pressure

Case No.	Patient	Sex	Age	Mean Arterial Blood Pressure Before Therapy mm. Hg.	After Therapy mm. Hg.	Decrease mm. Hg.
27	E.M.	M	48	153	135	18
28	R.W.	F	48	170	140	30
29	R.L.	F	47	290	110	180
30	V.A.	F	47	145	105	40
31	G.C.	M	46	185	118	67
32	J.T.	M	45	160	130	30
33	W.G.	M	45	200	125	75
34	E.H.	F	44	200	105	95
35	W.O.	M	42	160	100	60
36	B.C.	F	40	160	120	40
37	L.W.	F	36	205	145	60
38	F.L.	F	33	160	110	50
39	M.W.	F	32	165	128	37
40	A.D.	F	29	200	120	80

Average mean arterial blood pressure at beginning of therapy ..... 182 mm. Hg.
Average mean arterial blood pressure at end of therapy ..... 120 mm. Hg.
Average decrease in mean arterial blood pressure ..... 62 mm. Hg.
Mean arterial blood pressure = $\frac{\text{Systolic} + \text{Diastolic}}{2}$

Cases ranged from mild to severe hypertension, without crises, and no emergencies existed.

Seven patients in the series (17.5%) had never before responded to other types of antihypertensive agents.

Slight untoward reactions occurred in six patients (15%), but these were of a minor nature, some dizziness, which disappeared when the dosage was reduced.

Conclusions

The antihypertensive activity of a drug combination containing reserpine 0.1 mg., hydralazine 25 mg., and hydrochlorothiazide 15 mg. evidently is greater than the sum total of each drug when given alone in their established dosage range.

The drug combination under study contains sub-therapeutic doses when compared with the dose of each drug given independently. Therefore, the total daily intake of each of the three drugs in this compound is so small that it places the toxicity quotient and untoward reactions at their very minimum.

The therapeutic effects of this compound are these: reserpine provides the necessary calming and heart-slowing effects so often desirable in the treatment of hypertensives; hydralazine curtails the compensatory release of renal pressor substances by promoting blood flow to ischemic kidneys, thus eliminating a possible cause of elevated blood pressure; by relaxing cerebral vascular tone, hydralazine helps to relieve minor cerebral hypertensive symptoms; hydrochlorothiazide induces effective diuresis and reduces edema due to cardiac decompensation. This often eliminates the need for unpalatable salt-free diets.

Most hypertensives require more than one drug to control their blood pressure. Patients with moderate to severe hypertension, especially those with anxiety, relative renal or cerebral ischemia and/or cardiac decompensation may be readily managed on this compound, without any major therapeutic problems.

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Coming Meetings

University of Colorado Medical Center, Eighth Annual General Practice Review, Denver, Jan. 7-13, 1962.

University of Texas Postgraduate School of Medicine, El Paso Division, Postgraduate Course, El Paso County Medical Society Turner Home, 1301 Montana Avenue, Jan. 21, 1962.

International Medical Assembly of Southwest Texas, 26th Annual Session, Granada Hotel, San Antonio, Jan. 29-31, 1962.

District One, Texas Medical Association, Annual Meeting, Pecos Country Club, Pecos, Texas, Feb. 3, 1962.

American College of Allergists, Graduate Instructional Course and 18th Annual Congress, Hotel Radisson, Minneapolis, April 1-6, 1962.

Southwest Obstetrical and Gynecological Society, 12th Annual Meeting, Camelback Inn, Phoenix, Oct. 10-13, 1962.

Southwestern Medical Association, 44th Annual Meeting, Western Skies Hotel, Albuquerque, Oct. 18-20, 1962.

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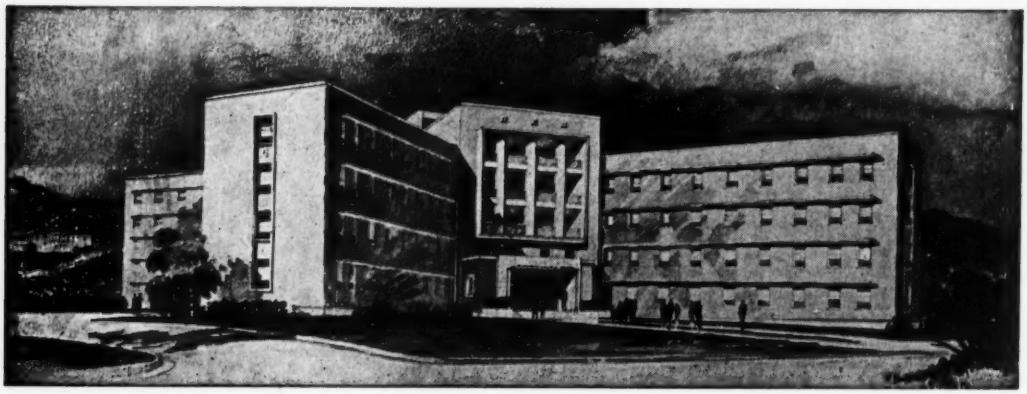

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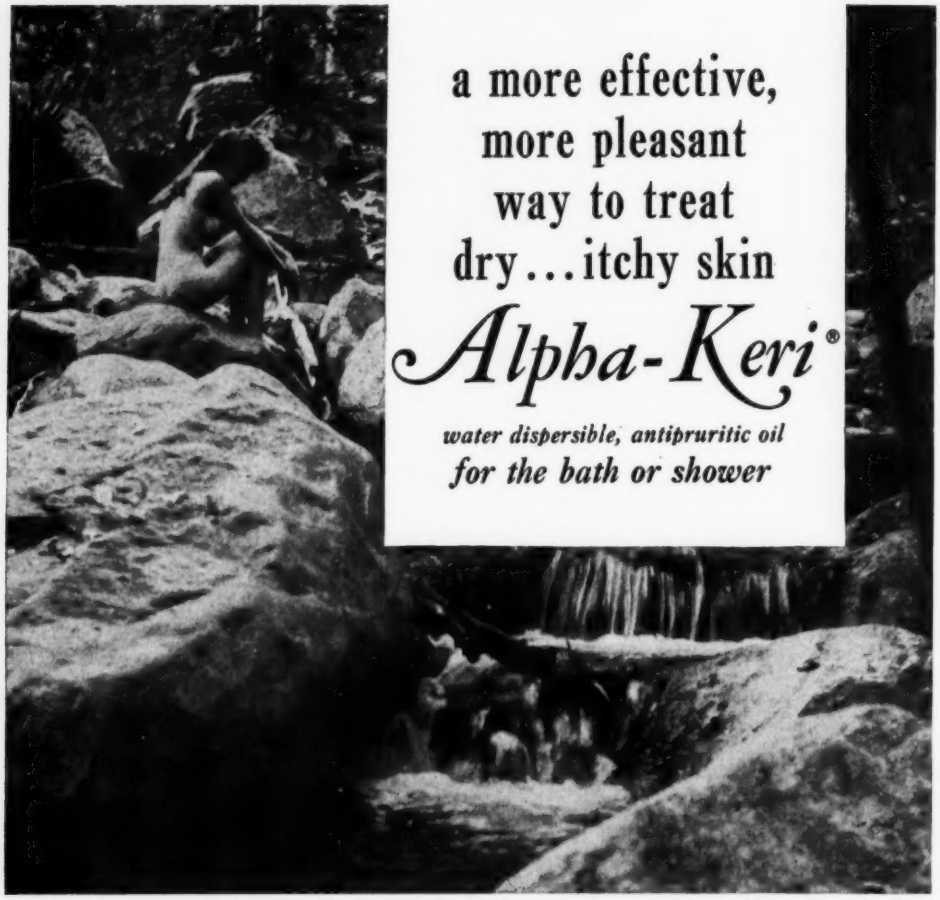

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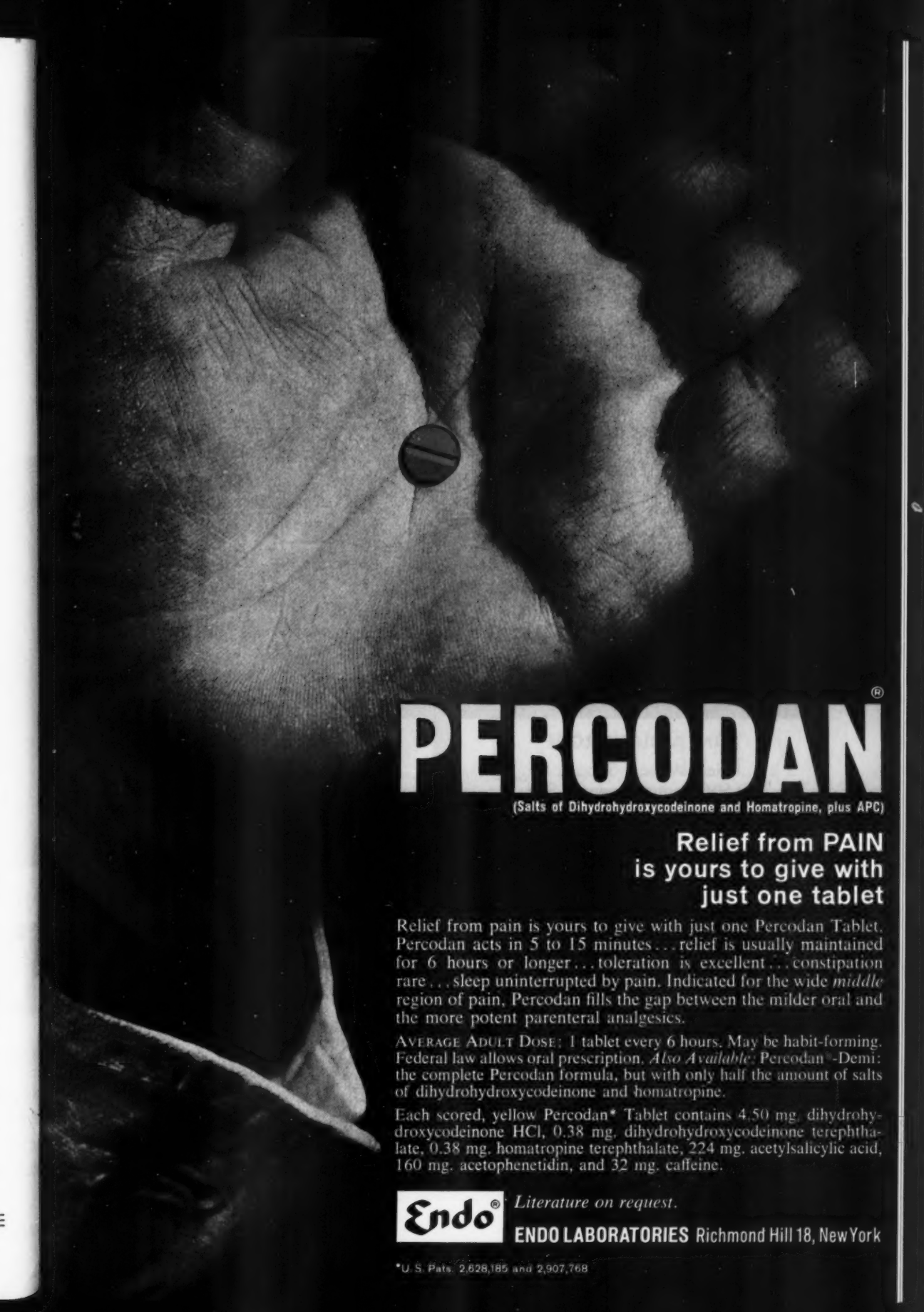

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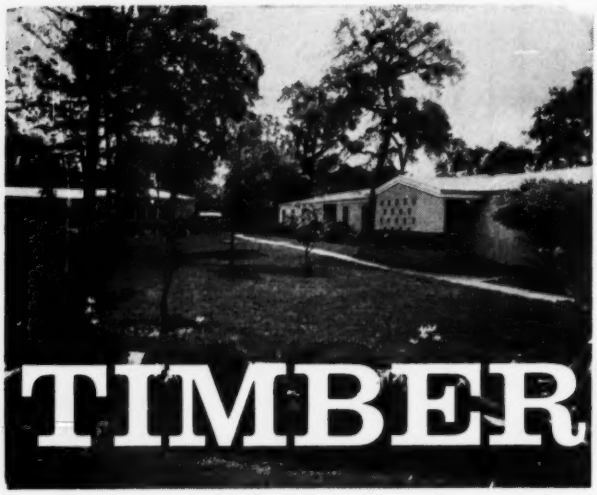

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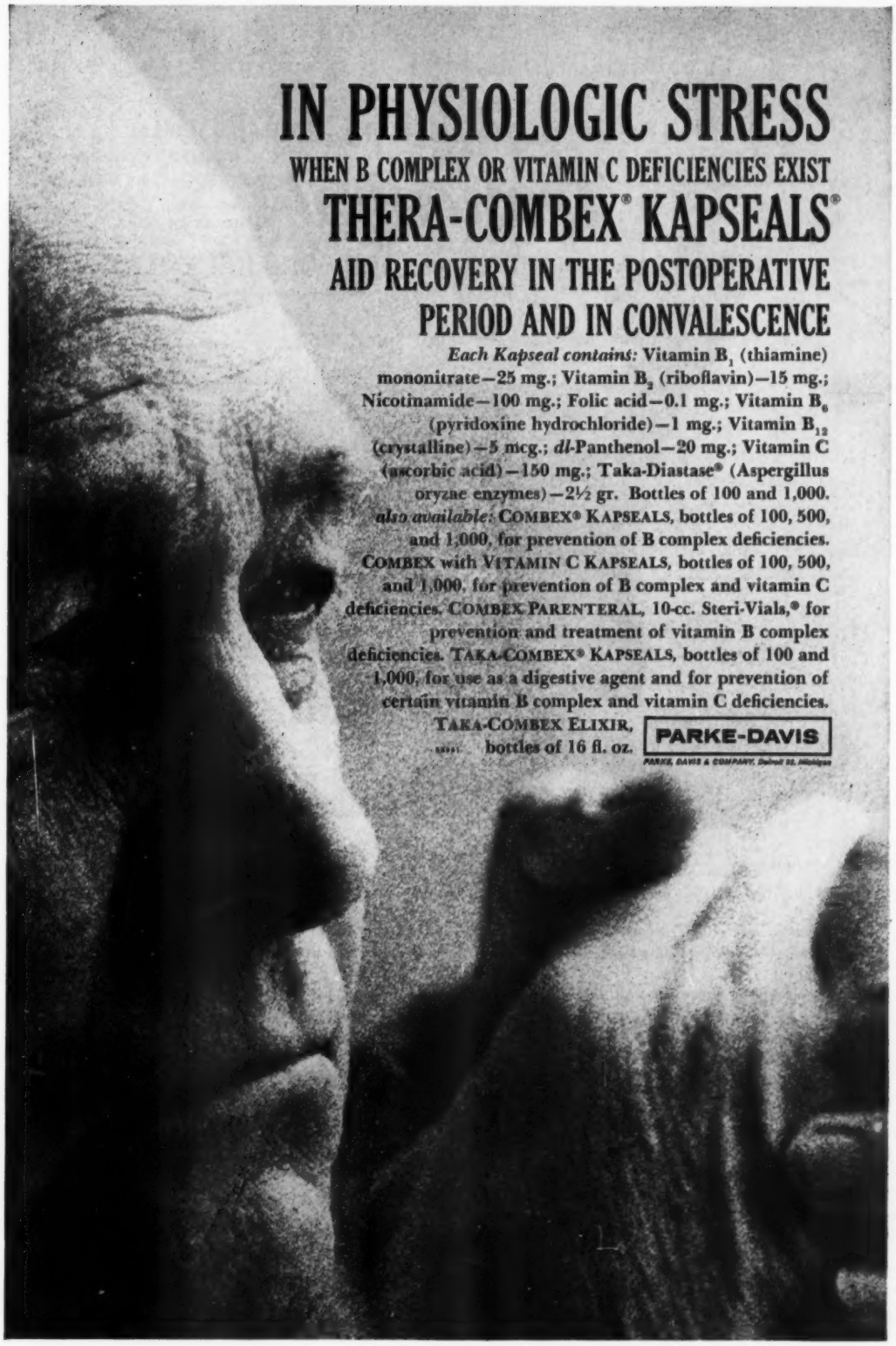
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References: 1. DeNyse, D. L.: M. Times 87:1512 (Nov.) 1959.
2. Gruenberg, F.: Current Therap. Res. 2:1 (Jan.) 1960.

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The first long-acting oral steroid, Medrol Medules gives the arthritic patient therapeutic action that continues through the night. In many cases, morning stiffness can become a thing of the past.

The slow, steady release of methylprednisolone often provides greater effectiveness, with less frequent administration and sometimes a reduced total daily dosage.

Many of your arthritic patients, too, can wake up comfortable on Medrol Medules.

Dosage: The following dosages are recommended in rheumatoid arthritis:

	Initial	*Maintenance*
Severe	12 to 16 mg.	6 to 12 mg.
Moderately severe	8 to 10 mg.	4 to 8 mg.
Moderate	6 to 8 mg.	2 to 6 mg.
Children	6 to 10 mg.	2 to 8 mg.

With Medrol Medules, it may be possible to reduce the total daily dose by ⅓.

Indications and effects: Medrol benefits (anti-inflammatory, antiallergic, antirheumatic, antileukemic, antihemolytic) have been demonstrated in acute rheumatic carditis, rheumatoid arthritis, asthma, hay fever and allergic disorders, dermatoses, blood dyscrasias, and ocular inflammatory disease involving the posterior segment.

Precautions and contraindications: Because of Medrol's high therapeutic ratio, patients usually experience dramatic relief without developing such possible steroid side effects as gastrointestinal intolerance, weight gain or weight loss, edema, hypertension, acne, or emotional imbalance.

As in all corticotherapy, however, there are certain cautions to be observed. The presence of diabetes, osteoporosis, chronic psychotic reactions, predisposition to thrombophlebitis, hypertension, congestive heart failure, renal insufficiency, or active tuberculosis necessitates careful control in the use of steroids. Like all corticosteroids, Medrol is contraindicated in patients with arrested tuberculosis, peptic ulcer, acute psychoses, Cushing's syndrome, herpes simplex keratitis, vaccinia, or varicella.

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Each capsule contains: Medrol (methylprednisolone) 2 mg. or 4 mg. Supplied in bottles of 30 and 100.

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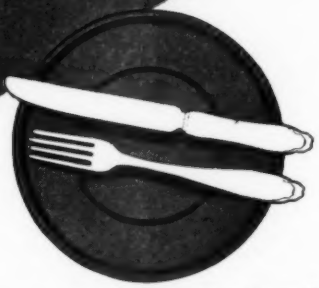

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for control of acute cough and associated allergic reactions

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for control of acute cough and for relief from associated muscular aches, pain and fever

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the threshold of the medullary cough center is elevated while the cough reflex is not abolished.

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inhibits tendency of histamine to cause edema of the nasopharyngeal mucosa, local irritation, and vasodilation.

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acetyl-p-aminophenol analgesic and antipyretic

FORMULAS:

ULO SYRUP—Each 5 ml. teaspoonful contains:

chlophedianol • HCl* [alpha-(2-dimethylaminoethyl)-o-chlorobenzhydrol • HCl]	25 mg.
chloroform, U.S.P.	0.001 ml.

Alcohol 6.65 per cent in a pleasant flavored syrup base

ULOMINIC® SYRUP — Each teaspoonful (5 cc) contains:

chlophedianol HCl* [alpha-(2-dimethylaminosthyl)-o-chlorobenzhydrol • HCl]	15.0 mg.
diphenylpyraline HCl (1-methyl-4-piperidyl-benzhydryl ether • HCl)	1.0 mg.
phenylephrine HCl	5.0 mg.
glyceryl guaiacolate	100.0 mg.
alcohol	6%

ULOGESIC® — Each tablet contains:

chlophedianol HCl* [alpha-(2-dimethylaminoethyl)-o-chlorobenzhydrol • HCl]	7.5 mg.
diphenylpyraline HCl (1-methyl-4-piperidyl-benzhydryl ether • HCl)	0.5 mg.
phenylephrine HCl	2.5 mg.
glyceryl guaiacolate	25.0 mg.
acetaminophen	162.5 mg.

*Patents pending

INDICATIONS: For acute cough associated with:

Upper Respiratory Infections
Common Cold
Influenza
Pneumonia
Allergies (Ulominic and Ulogesic)
Pertussis
Pleurisy
Bronchitis
Tracheitis
Laryngitis
Croup

CONTRAINDICATIONS: Although no contraindications for ULOMINIC or ULOGESIC are known, they should be used only for acute cough.

CAUTION: Since ULOMINIC and ULOGESIC contain an antihistaminic agent, drowsiness may occur. As they also contain a sympathomimetic agent, they should be used with caution in coronary artery disease, glaucoma, hypertension, and hyperthyroidism.

SIDE EFFECTS:

ULO
These occur only occasionally and have been mild. Nausea and dizziness have occurred infrequently; vomiting and drowsiness rarely. As with all centrally acting drugs, an infrequent case may develop excitation, hyperirritability and nightmares. The symptoms disappear within a few hours after the drug is discontinued. In three cases (1 adult and 2 children) where the drug was continued in large or even excessive amounts after stimulation was present, hallucinations developed. Upon withdrawal of the medication, the patients recovered rapidly within a few hours.

ULOMINIC and ULOGESIC
Side effects from ULOMINIC or ULOGESIC occur occasionally and are mild. Nausea, dizziness, and dryness of the mouth occur infrequently; vomiting and drowsiness rarely.

DOSAGE:

ULO
Adults: 25 mg. (1 teaspoonful) 3 or 4 times daily as required.
Children: 6 to 12 years of age—12.5 to 25 mg. (½ to 1 teaspoonful) 3 or 4 times daily as required;
2 to 6 years of age—12.5 mg. (½ teaspoonful) 3 or 4 times daily as required.

ULOMINIC
Adults: One teaspoonful (5 cc) four times daily.
Children: 6 to 12 years—½ teaspoonful (2.5 cc) 4 times daily.
2 to 6 years—¼ teaspoonful (25 drops) 4 times daily.

ULOGESIC
Adults: Two tablets 4 times daily.
Children: 6 to 12 years—one tablet 4 times daily.

AVAILABILITY:

ULO SYRUP
Bottles 12 oz.

ULOMINIC SYRUP
Bottles 1 pint

ULOGESIC TABLETS
Bottles of 100 tablets.

CAUTION: Federal Law prohibits dispensing without prescription.

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